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Therapeutic effect of vitamin D supplementation on glycemic control in female patients with type 2 diabetes mellitus: A randomized controlled trial

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Abstract: This randomized controlled trial examined the relationship between therapeutic doses of vitamin D and improvement of glycemic parameters in female patients with Type 2 Diabetes Mellitus (T2DM). 27 female Libyan patients with T2DM, aged 38 - 64 years, were randomly assigned to the intervention group (n = 15) and the control group (n = 12) at the endocrinology department of Tripoli Medical Center from 2015 to 2023. The intervention group received 50,000 IU vitamin D (cholecalciferol) weekly for 12 weeks, while controls received a placebo. Serum levels of 25-hydroxyvitamin D [25(OH)D], fasting blood glucose, HbA1c, lipid profile, and calcium were measured before and after intervention. Data were analyzed using paired t-test and correlation analyses. Baseline vitamin D deficiency was prevalent: 47.0% of patients had severe deficiency, 41.0% moderate deficiency, and 11.0% had sufficient levels. After 12 weeks of supplementation, 88.0% of patients demonstrated improved vitamin D levels. Mean serum 25(OH)D increased significantly from 12.50 ± 12.40 nmol/L to 37.00 ± 10.00 nmol/L. About 80.0% of patients exhibited decreased HbA1c from 9.20 ± 1.80% to 7.90 ± 1.70%; $p < 0.05$. A significant inverse correlation was observed between post-intervention HbA1c and 25(OH)D levels ($r = -0.354$, $p = 0.04$). Fasting blood glucose improved from 174.44 ± 63.77 mg/dL to 144.51 ± 38.90 mg/dL. No significant changes occurred in the control group. Weekly vitamin D supplementation for 12 weeks significantly improved vitamin D status and glycemic control in female patients with T2DM, demonstrating an inverse relationship between vitamin D and HbA1c levels. Vitamin D may be a beneficial adjunct therapy for glycemic management in vitamin D-deficient diabetic patients.

Introduction

Type 2 Diabetes Mellitus (T2DM) is one of the most prevalent chronic metabolic disorders worldwide and represents a major global health challenge. According to the International Diabetes Federation, more than 537 million adults were living with diabetes in 2021, and this number is expected to increase substantially in the coming decades if effective preventive strategies are not implemented [1]. T2DM is characterized by chronic

hyperglycemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction [2, 3]. Persistent hyperglycemia is associated with long-term complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy, which contribute significantly to morbidity and mortality among diabetic patients. Several environmental and lifestyle factors contribute to the development and progression of T2DM. These include obesity, sedentary lifestyle, unhealthy dietary patterns, cigarette smoking, and genetic predisposition [4-9]. In recent years, increasing attention has been directed toward micronutrient deficiencies as potential contributors to metabolic disorders. Among these, vitamin D deficiency has emerged as a possible modifiable risk factor associated with impaired glucose metabolism and increased risk of T2DM [10, 11].

Vitamin D is a fat-soluble secosteroid hormone primarily synthesized in the skin after exposure to ultraviolet B radiation, although smaller amounts can be obtained from dietary sources such as fatty fish, fortified dairy products, and supplements. After synthesis or ingestion, vitamin D undergoes hydroxylation in the liver to form 25-hydroxyvitamin D [25(OH)D], which is the major circulating form and the most reliable indicator of vitamin D status [12]. A second hydroxylation step occurs in the kidneys to produce the biologically active form, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D]. Accumulating evidence suggests that vitamin D plays an important role in glucose homeostasis and insulin metabolism. Vitamin D receptors and the enzyme 1-α-hydroxylase are expressed in pancreatic β-cells and in insulin-sensitive tissues such as skeletal muscle and adipose tissue [13, 14]. Through these receptors, vitamin D may enhance insulin secretion, improve insulin sensitivity, and regulate inflammatory processes that contribute to insulin resistance [15]. In addition, vitamin D influences intracellular calcium regulation in pancreatic β-cells, which is essential for insulin secretion [16].

Observational studies have reported that individuals with low serum 25(OH)D concentrations are more likely to develop impaired glucose tolerance, metabolic syndrome, and T2DM [11, 17]. Several epidemiological studies have also demonstrated an inverse relationship between vitamin D levels and glycemic indicators such as fasting blood glucose and glycated hemoglobin (HbA1c) [17, 18]. Furthermore, vitamin D deficiency has been reported to be highly prevalent among patients with T2DM, particularly in Middle Eastern and North African populations [19]. Despite the growing evidence suggesting a relationship between vitamin D status and glucose metabolism, clinical trials evaluating the therapeutic effects of vitamin D supplementation on glycemic control have produced inconsistent results. While some randomized controlled trials have reported significant improvements [20, 21], others have observed minimal or no effects [22]. These inconsistencies may be attributed to differences in study design, baseline vitamin D status, dosage, duration of supplementation, and population characteristics. Given the high prevalence of T2DM and vitamin D deficiency in the Middle East and North Africa region, further investigation is warranted. However, data from Libya and neighboring countries remain limited. Therefore, the present randomized controlled trial aimed to evaluate the effect of vitamin D supplementation (cholecalciferol 50,000 IU weekly) on serum vitamin D levels and glycemic parameters in female patients with T2DM. Additionally, the study examined the relationship between serum 25(OH)D concentrations and indicators of glycemic control.

Materials and methods

Study design and participants: This was a randomized, double-blind, placebo-controlled clinical trial conducted at the endocrinology department of Tripoli Medical Center, Libya, from 2015 to 2023. A total of 27 female Libyan patients with T2DM, aged 38 to 64 years, were enrolled in the study.

Inclusion criteria: Female patients diagnosed with T2DM for at least five years, aged between 38 and 64 years, with no history of renal or hepatic diseases based on medical records, no other significant underlying health conditions, and no use of calcium or vitamin D supplements within the six months before participation.

Exclusion criteria: Included male patients, individuals with type 1 diabetes mellitus, pregnant or lactating women, those with renal or hepatic impairment, current users of vitamin D or calcium supplements, and patients with a history of parathyroid disorders.

Randomization and intervention: Participants were randomly assigned to two groups using a computer-generated random allocation sequence. Allocation concealment was ensured by sealed, opaque envelopes that were opened only after participant enrollment. Intervention group (n = 15): Received oral vitamin D3 (cholecalciferol) 50,000 IU tablet once weekly for 12 weeks. Control group (n = 12): Received placebo tablets once weekly for 12 weeks, matching the duration of the intervention group (to mitigate risks associated with no treatment).

Laboratory measurements: Blood samples were collected from all participants using red and grey vacutainers. Red-capped vacutainers were used for estimating total plasma vitamin D [25(OH)D], HbA1c, lipid profile (total cholesterol, triglycerides, HDL, LDL), and calcium levels. Grey-capped vacutainers were used for fasting blood glucose (FBG) estimation after an overnight fast. All parameters were assessed using the Cobas 6000 analyzer series before and after the 12-week intervention period.

Ethical considerations: Oral informed consent was obtained from all patients before participation. The approval was obtained from the Ethical Review Committee of the Tripoli Medical Center, Tripoli, Libya (Reference No. 066-MJ-2025 and TMC, 4/26/2025).

Statistical analysis: Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20. Descriptive statistics, including mean and standard deviation (SD), were employed to summarize the data. Paired t-tests were used to evaluate the significance of changes within groups before and after intervention. Correlation tests (Pearson's correlation coefficient) were conducted to assess relationships between variables. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 27 female patients with T2DM were enrolled, with a mean age ranging from 38 to 64 years. Baseline assessment revealed that 47.0% of diabetic patients had severe vitamin D deficiency, 41.0% had moderate deficiency, and 11.0% had sufficient vitamin D levels. Following 12 weeks of vitamin D supplementation, 88.0% of patients in the intervention group showed improvement in their vitamin D levels. Serum 25(OH)D increased significantly from 12.50 ± 12.40 nmol/L at baseline to 37.0 ± 10.0 nmol/L post-intervention (**Figure 1)**.

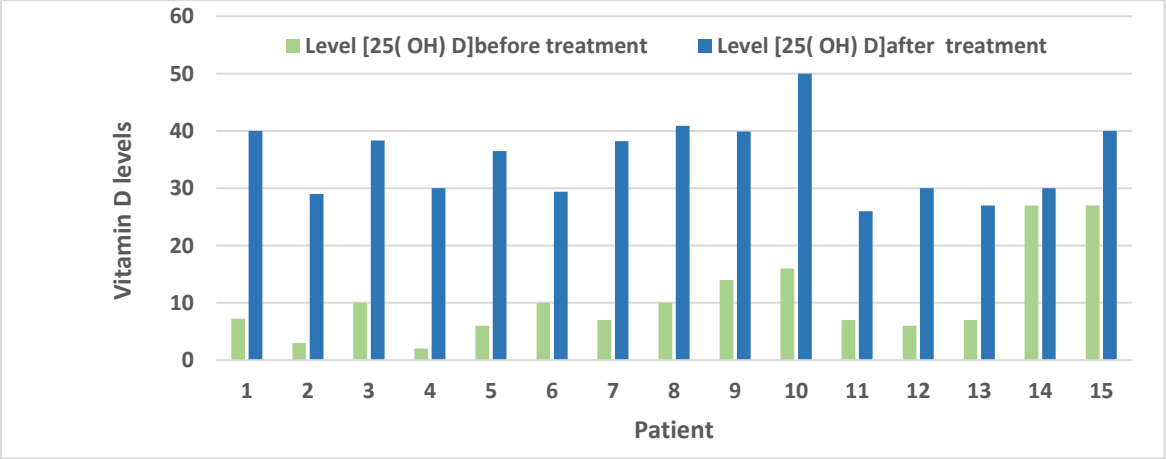

Figure 1: Vitamin D levels before and after 12 weeks of supplementation
A bar graph showing mean 25(OH)D levels increasing from 12.5 nmol/L to 37.0 nmol/L.

Changes in glycemic parameters: Around 80.0% of patients in the intervention group exhibited a significant decrease in HbA1c after treatment. Mean HbA1c decreased significantly from 9.20 ± 1.80% at baseline to 7.90 ± 1.70% post-intervention ($p < 0.05$) (**Table 1, Figure 2**). Fasting blood glucose also showed improvement, decreasing from 174.44 ± 63.77 mg/dL to 144.51 ± 38.90 mg/dL.

Table 1: Glycemic parameters before and after intervention

Parameter	Before treatment (mean ± SD)	After treatment (mean ± SD)	p-value
HbA1c (%)	9.20 ± 1.80	7.90 ± 1.70	< 0.05
FBG (mg/dL)	174.44 ± 63.77	144.51 ± 38.90	< 0.05

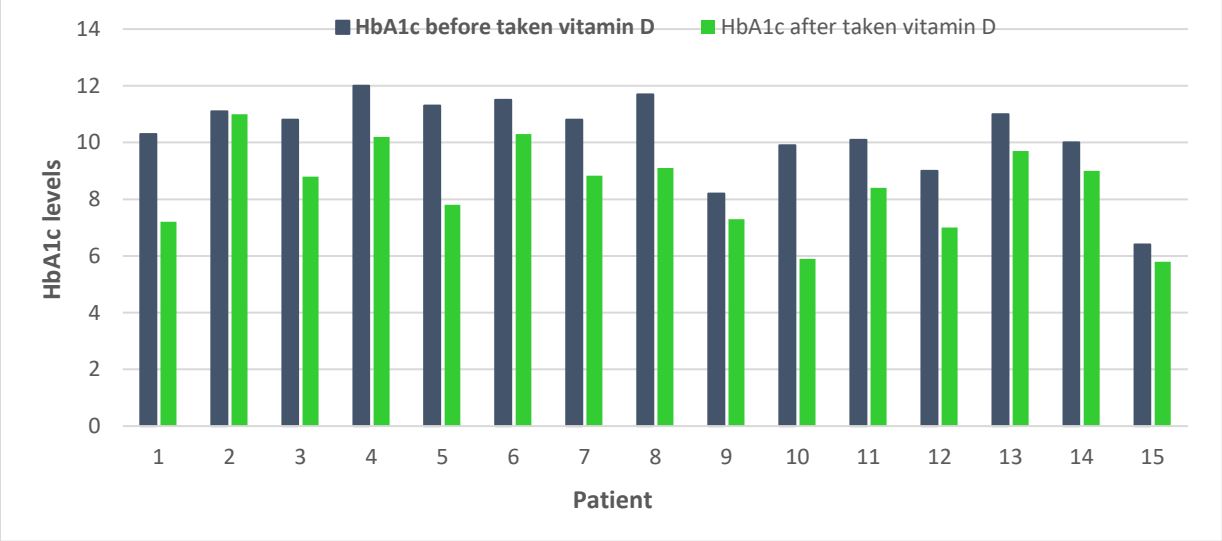

Figure 2: HbA1c levels before and after 12 weeks of vitamin D supplementation
A bar graph showing mean HbA1c decreasing from 9.2% to 7.9%.

Correlation between vitamin D and glycemic control: A significant inverse correlation was observed between post-intervention HbA1c and 25(OH)D levels ($r = -0.354$, $p = 0.040$), indicating that higher vitamin D levels were associated with better glycemic control.

Changes in lipid profile and calcium: Vitamin D supplementation resulted in modest improvements in lipid parameters and a significant increase in serum calcium levels (from 8.21 ± 1.25 mg/dL to 9.24 ± 0.90 mg/dL; $p < 0.05$) (**Table 2**).

Table 2: Lipid profile and calcium levels before and after intervention

Parameters	Before treatment (mean ± SD)	After treatment (mean ± SD)
Total cholesterol (mg/dL)	177.49 ± 33.88	161.99 ± 30.57
Triglycerides (mg/dL)	150.0 ± 80.44	116.63 ± 48.55
HDL (mg/dL)	50.96 ± 21.04	47.74 ± 15.76
LDL (mg/dL)	133.25 ± 108.69	105.28 ± 36.09
Calcium (mg/dL)	8.21 ± 1.25	9.24 ± 0.90

Control group findings: No significant changes were observed in any measured parameters. Mean HbA1c remained relatively unchanged (7.98 ± 1.67% to 8.14 ± 1.68%), and vitamin D levels showed no significant improvement (10.08 ± 6.66 nmol/L to 9.97 ± 6.62 nmol/L) (**Table 3**).

Table 3: Control group parameters before and after placebo

Parameter	Before placebo (mean ± SD)	After placebo (mean ± SD)	p-value
HbA1c (%)	7.98 ± 1.67	8.14 ± 1.68	NS
25(OH)D (nmol/L)	10.08 ± 6.66	9.97 ± 6.62	NS
FBG (mg/dL)	154.5 ± 71.34	156.6 ± 70.99	NS

NS = not significant

Discussion

The primary objective of this study was to evaluate the effect of vitamin D supplementation on glycemic parameters in female patients with T2DM. The results demonstrated a significant inverse relationship between vitamin D levels and HbA1c, suggesting that maintaining adequate vitamin D status may contribute to improved glycemic control. This finding is consistent with previous evidence highlighting the potential role of vitamin D in glucose metabolism and diabetes management [1, 23-27]. The present randomized controlled trial showed that weekly cholecalciferol supplementation over 12 weeks significantly increased serum 25-hydroxyvitamin D levels. More importantly, a significant reduction in HbA1c and fasting blood glucose was observed in the intervention group, alongside a clear inverse correlation between vitamin D status and HbA1c. Similar improvements in glycemic control have been reported in other clinical trials and meta-analyses, supporting the beneficial effect of correcting vitamin D deficiency in patients with T2DM [5, 23, 28].

Vitamin D deficiency was highly prevalent among participants at baseline, with most showing moderate to severe deficiency. This aligns with previous reports indicating a high prevalence of vitamin D deficiency in individuals with T2DM, particularly in Middle Eastern populations, despite abundant sunlight exposure. Contributing factors include limited sun exposure, clothing practices, and dietary insufficiency, with reported deficiency rates reaching 60.0-90.0% in this region [11, 29]. The observed reduction in HbA1c is clinically significant, as even small decreases in HbA1c are associated with reduced risk of diabetic complications. Prior studies and meta-analyses have similarly shown that vitamin D supplementation can lead to modest but meaningful improvements in glycemic markers, particularly in deficient individuals [16, 28]. Several biological mechanisms may explain these effects. Vitamin D receptors are present in pancreatic β-cells, skeletal muscle, and adipose tissue, indicating a role in glucose homeostasis. Vitamin D enhances insulin secretion by regulating intracellular calcium in β-cells and may improve insulin sensitivity in peripheral tissues [11, 13]. Additionally, vitamin D exerts anti-inflammatory effects by suppressing pro-inflammatory cytokines such as TNF-α and IL-6, thereby reducing insulin resistance [24, 30]. Another proposed mechanism involves modulation of the renin-angiotensin system, where vitamin D suppresses renin expression, potentially improving β-cell function and insulin sensitivity [21]. These combined effects may explain the improvement in glycemic control observed in this study. Although lipid profile changes were not statistically significant, slight improvements were observed. Previous research suggests vitamin D may influence lipid metabolism indirectly through improved insulin sensitivity and reduced inflammation, potentially contributing to cardiometabolic benefits [11, 31]. Despite these findings, limitations should be acknowledged. The relatively small sample size and inclusion of only female participants limit generalizability. Additionally, the short intervention duration (12 weeks) may not fully reflect long-term metabolic effects. Other influencing factors, such as sun exposure, diet, and physical activity, were not controlled, which may affect vitamin D status and metabolic outcomes [20, 30]. Future research should focus on larger multicenter randomized trials including both genders and longer follow-up periods to confirm these findings. Determining optimal dosing strategies and identifying populations most likely to benefit from supplementation remain important areas for further investigation. Overall, this study adds to the growing body of evidence suggesting that vitamin D supplementation may serve as a beneficial adjunctive strategy for improving glycemic control in patients with T2DM.

Conclusion: This randomized controlled trial demonstrates that weekly supplementation with 50,000 IU vitamin D for 12 weeks significantly improves vitamin D status and glycemic control in female patients with T2DM. The significant inverse correlation between vitamin D and HbA1c levels suggests that vitamin D plays a beneficial role in glucose homeostasis. Given the high prevalence of vitamin D deficiency among diabetic patients in Libya, routine screening for vitamin D deficiency and appropriate supplementation should be considered as part of comprehensive diabetes management.

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Data availability statement: The raw data that support the findings of this article are available from the corresponding author upon reasonable request.
Author declarations: The authors confirm that they have followed all relevant ethical guidelines and obtained any necessary IRB and/or ethics committee approvals.
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